

**IN THE UNITED STATES PATENT AND TRADEMARK OFFICE**

Applicant(s): Houghton, et al.	
Application No.: 09/996,128	
Filed: 11/27/2001	Group Art Unit: 1643
Title: Compositions for Treatment of Melanoma and Method of Using Same	Examiner: A Harris
Attorney Docket No.: MSK.P-026-3	

**BRIEF FOR APPELLANT**

This brief is filed in support of Applicants' Appeal from the final rejection mailed 12/27/2006. Consideration of the application and reversal of the rejections are respectfully urged.

Real Party in Interest

The real parties in interest are Sloan-Kettering Institute for Cancer Research and The Animal Medical Center.

Related Appeals and Interferences

To Applicant's knowledge there are no related Appeals or Interferences.

Status of Claims

Claims 20-27, 29 and 30 are pending in the application. Claims 1-19 and 28 have been canceled. Claim 24 is allowed. Claims 20-23, 29 and 30 are rejected and are the subject of this appeal. Claims 25-27 are withdrawn from consideration.

### Status of Amendments

The amendment after final filed on 2/27/2007 has been entered (Advisory Action of 7/3/2007).

### Summary of Claimed Subject Matter

As reflected in independent claim 20, the claims of the present application relate to a method for treatment of canine malignant melanoma in a dog suffering from canine malignant melanoma. Canine malignant melanoma (CMM) of the oral cavity, nail bed and mucocutaneous junction is a spontaneously occurring, highly aggressive metastatic neoplasm. CMM is initially treated with aggressive surgery and/or fractionated radiation therapy, however systemic metastatic disease is quite common and invariably chemoresistant. (Page 11, lines 19-22) In accordance with the method of the invention, an immunologically-effective amount of a xenogeneic differentiation antigen of the same type as a differentiation antigen expressed by melanoma cells of the dog is administered to the dog (Page 2, lines 22-27; Page 3, lines 22-26) . As shown in Example 7 (Page 11, line 27-Page 12, line 27) the treatment resulted in prolonged life (over that expected for dogs with CMM) and in one of nine dogs treated a complete response from Stage IV cancer.

### Grounds of Rejection to be reviewed on Appeal

Claims 20-23, 29 and 30 stand rejected under 35 USC § 103 as obvious over Zhai et al, in view of US Patent No. 5,773,291 and US Patent No. 6,080,727.

### Argument

Claims 20-23, 29 and 30 stand rejected under 35 USC § 103 as obvious over Zhjai et al, in view of US Patent No. 5,773,291 and US Patent No. 6,080,727. Applicants submit that this rejection is in error for several reasons, and argues several groups of claims below for separate consideration.

Independent Claim 20 and claims 23 and 29 are not obvious over the cited art

Zhai, the principle reference in this case, relates to inducement of T cell immunity using recombinant adenovirus encoding melanoma tumor-associated antigens MART1 or gp100 for use in cancer therapy. On Page 705 of the Zhai reference, there is a teaching that mice immunized with adenovirus encoding human MART1 or gp100 were protected from tumor challenge with B16 murine melanoma cells. In rejecting claim 20 as obvious over the Zhai, the Examiner has treated this teaching of one type of melanoma as a teaching of all types of melanoma, including canine malignant melanoma as recited in claim 20. With respect to inducing the immune response in dogs instead of mice, the Examiner has cited US 6,080,727. Applicants submit that these teaching do not render the claimed invention obvious.

Claim 20 does not relate to melanoma in general, but to a specific condition: canine malignant melanoma (CMM). CMM is known to be very aggressive, malignant and metastatic. For example, in 1999, Modiano et al. stated that "Canine malignant melanoma is a rapidly metastatic disease that generally is incurable." (See Exhibit Appendix) In 2006, investigators at the University of California at Davis stated that "the fatality rate of this cancer is very high despite aggressive treatment with surgery, radiation therapy and chemotherapy." (See, Exhibit Appendix) Similarly, the National Canine Cancer Foundation web site <http://www.wearethecure.org/melanoma.htm> observes that

Melanoma occurs commonly in dogs with pigmented (dark) skin. Melanomas arise from pigment producing cells called melanocytes, which are responsible for coloring the skin. Any dog can be affected, but Gordon Setters, Standard and Miniature Schnauzers, Doberman Pinschers, and Scottish terriers, among others, are at increased risk to develop melanoma, suggesting that this disease may have a hereditary component. Melanomas can occur in areas of haired skin, where they usually form small, dark (brown to black) lumps, but can also appear as large, flat, wrinkled masses. Melanoma of the haired skin in dogs is usually a benign tumor, although it can cause severe discomfort. **In contrast, malignant melanoma, which develops in the mouth or in the distal limbs (usually the toenail beds), is an incurable disease.** These tumors have very often spread to distant parts of the body (metastasized) by the time they are first noticed, making complete surgical removal impossible.

Thus, CMM is known to be different from other melanomas, including other melanomas in dogs, in its treatability and response to therapy.

In the final office action mailed December 27, 2006, the Examiner asserted (without support) that Zhai shows induction of T cell immunity to mammalian (murine) metastatic melanoma. (Page 4). In a responsive declaration, this statement concerning the B16 murine melanoma was shown to be untrue. Specifically, in the declaration (copy attached in the Evidence Appendix), it is stated that B16 melanoma is not per se metastatic, and the parental B16 melanoma cells rarely metastasize. (Declaration, ¶ 3) The Zhai et al paper reports only B16 melanoma, and do not describe a metastatic derivative. Further, the tests performed in Zhai et al. have nothing to do with assessment of metastasis. (Declaration, ¶ 4).

Moreover, the declaration observes that because B16 melanoma likely arose from a different source than the mucosal origin of CMM and is therefore not a viable model for CMM. Furthermore, because B16 responds to drugs that are inactive against cutaneous and mucosal melanomas, it is not apparent that results for B16 provides any expectation of success in the treatment of CMM. (Declaration, ¶ 5).

In response to this argument, the Examiner did not provide any evidence that B16 melanoma is metastatic, or that it provides a valid model for CMM which arises from a different cell type from the B16 cell line. Rather, the Examiner simply maintains the argument in the face of the evidence to the contrary that Zhai et al has something to do with treatment of a metastatic tumor, and is applicable to CMM. This alone should be a basis for reversal of the rejection.

The Examiner also states that "applicant is reminded that metastatic" is not a limitation recited in claim 20. (Advisory Action, 7/3/07) This argument is irrelevant because "canine malignant melanoma" is recited in claim 20, and high rate of metastasis is an inherent characteristic of this disease as reflected by the evidence of record.

Furthermore, the Examiner's citation of US 6,080,727 to bridge the gap between the mice of Zhai and the dogs treated in the present invention is a patchwork hindsight reconstruction that is not supported by the references. The '727 patent can at best be said to provide support for the proposition that melanoma (as a general term) occurs in dogs, but since the reference has nothing

to do with melanoma differentiation antigens (the antisense used in this reference is to the c-myc sequence which is unrelated to any melanoma-specific antigen) nothing further can be reasonably extracted from this reference.

Thus, the Examiner's argument is essentially that because an easily treatable form of mouse melanoma can be treated with adenovirus encoding human MART1 or gp100, it would have been obvious that treating a very different form of highly aggressive in dogs would have been expected to succeed because it is known that some forms of melanoma occur in dogs and can be treated with a wholly unrelated oligonucleotide. This argument does not establish a *prima facie* case of obviousness, nor does it take into account the surprising fact that treatment efficiency is observed against this very aggressive disease that has been considered to be "generally ...incurable."

The rejection of claims 20, 23 and 29 should therefore be reversed.

#### Claims 21 and 22 are not Obvious

Dependent claim 21 specifies that the xenogeneic melanoma-associated differentiation antigen is tyrosinase, while dependent claim 22 specifies that it is human tyrosinase. Both of these claims are dependent on claim 20, and therefore are patentable for all of the reasons discussed above.

In addition, the primary Zhai reference does not teach anything with respect to tyrosinase. The Examiner has cited US Patent No. 5,773,291 as teaching expression of human tyrosinase within a vector, and argues that using tyrosinase in place of MART1 or gp100 in Zhai would have been obvious. In the non-final action mailed July 18, 2006, the Examiner states that the reason this substitution would have been obvious is "because it is well known in the art that tyrosinase ..., quite like gp100 is recognized as a TAA (tumor associated antigen in the development of cancer vaccines and the Zhai treatment was advantageous." The basis for this statement is the identification in the first introductory paragraph of the Zhai et al. paper of tyrosinase as one of several identified genes encoding tumor-associated antigens. there is a

marked lack of congruence between this speculation concerning opportunities and the Examiner's statement of what is "well-known" and what is reasonably likely to be successful. Furthermore, the Zhai paper then lists additional characteristics of MART1 and gp100 including involvement with tumor regression, and says that these characteristics (which are not attributed to tyrosinase) make them "excellent candidates for the development of Ag-specific vaccines." No such encouraging statement is made with tyrosinase, and therefore Applicants submit that the Examiner is finding the expectation of success in the present application rather than in the cited art.

Accordingly, Applicants submit that claims 21 and 22 are separately patentable over the cited references for these additional reasons.

Claim 30 is not obvious over the cited art

Claim 30 is dependent on claim 20, and recites the additional limitation that the xenogeneic differentiation antigen is administered by DNA immunization of the subject with DNA encoding the xenogeneic differentiation antigen in a **non-viral plasmid vector** comprising DNA encoding the xenogeneic differentiation antigen under the control of a promoter which promotes expression of the xenogeneic differentiation antigen.

Thus, this claim differs further from the teaching of Zhai et al in the nature of the vector being employed, since Zhai et al use an adenoviral vector. This difference is of patentable significance.

The Examiner has taken the position, without evidentiary support, that one vector is much like another. Applicants have argued that this is not the case, and the Examiner has offered no contrary evidence. The present application presents clear evidence that treatment with self-antigens as opposed to xenogeneic antigens are not generally useful even in the case of B16 melanoma, the same melanoma cell line used in the Zhai reference. (Pages 6-7, Examples 1 and 23). This effect cannot be predicted from the teaching of Zhai et al, however, because Zhai uses adenoviral vectors. Adenoviral vectors (and also vaccinia viral vectors) are known for their ability to act as adjuvants to produce an immune response, even where the antigen is not

generally useful. Thus, even the self gene expressed in adenovirus can give immunity to cancer. For instance, Perricone et al. (Molecular Therapy 1:275-284, 2000) show that adenovirus vectors expressing either human and mouse gp100 inhibit tumor growth in mice. In addition, Overwijk et al. (PNAS 96:2092-2097, 1999) showed that vaccinia vector (a viral vector with similar properties) expressing mouse TRP-1 (also called TYRP1, gp75) inhibited mouse melanoma. Thus, in both cases the virus encoding the self gene inhibited growth of a mouse tumor.

The Zhai et al. manuscript shows xenogeneic gp100 works when delivered by the adenovirus, but this is a special type of vaccine (with substantial risks for use clinically) that also works for self-antigen. Because of this knowledge of the adjuvant activity of the adenoviral vector employed by Zhai, the person skilled in the art could not separate the effect of the vector employed from those of the xenogeneic antigen to arrive at a conclusion that the xenogeneic antigen would be effective in a non-viral vector. For plasmid DNA vaccines (non-viral), DNA vaccination with the self-antigen does not result in tumor rejection. One needs the xenogeneic non-self DNA. (Examples 2 and 5, Fig. 5).

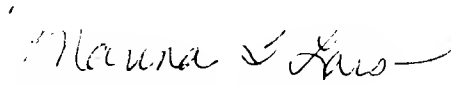
Thus, for this additional reason the application of Zhai to the claims in general, and to claim 30 in particular (which recites a non-viral plasmid vector) is not supportive of an obviousness rejection.

## Conclusion

In rejecting the claims of this application, the Examiner has relied on isolated facts from the references as a basis for the rejection, and has neglected to give weight to the overall state of the art and the knowledge that a skilled person would have taken into account in considering what was and what was not obvious. She has over-simplified circumstances by considering that one easy to deal with lab strain of melanoma cell line is equivalent to the generally incurable canine malignant melanoma simply because both include the term melanoma, notwithstanding declaration evidence to the contrary. Further she has taken the simplistic view that one antigen and one vector are the same as any other antigen vector without any scientific reasoning or

support in the face of substantial arguments and evidence to the contrary. In short, the rejection of the claims in this case fly in the face of applicable legal standards for an obviousness rejection, and they should be reversed.

Respectfully submitted,

A handwritten signature in cursive script, appearing to read "Marina T. Larson", written in black ink.

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Marina T. Larson Ph.D.  
PTO Reg. No. 32,038  
Attorney for Applicant  
(970) 262 1800



## **Claims Appendix**

20. A method for treating canine malignant melanoma in a dog suffering from canine malignant melanoma comprising administering to the dog an immunologically-effective amount of a xenogeneic differentiation antigen of the same type as a differentiation antigen expressed by melanoma cells of the dog.

21. The method according to claim 20, wherein the xenogeneic melanoma-associated differentiation antigen is tyrosinase.

22. The method according to claim 20, wherein the xenogeneic melanoma-associated differentiation antigen is human tyrosinase.

23. The method according to claim 20, wherein the xenogeneic melanoma-associated differentiation antigen is administered as a vector comprising a DNA sequence encoding the xenogeneic therapeutic melanoma-associated differentiation antigen under the control of a promoter which promotes expression of the xenogeneic melanoma-associated differentiation antigen in the dog.

29. The method of claim 20, wherein the differentiation antigen is selected from the group consisting of Melan-A/Mart-1, Pmel17, tyrosinase and gp75.

30. The method of claim 20, wherein the xenogeneic differentiation antigen is administered by DNA immunization of the subject with DNA encoding the xenogeneic differentiation antigen in a non-viral plasmid vector comprising DNA encoding the xenogeneic differentiation antigen under the control of a promoter which promotes expression of the xenogeneic differentiation antigen.

## **Evidence Appendix**

Declaration Under Rule 132 filed 2/27/2007

Modiano 1999 (attached to amendment filed 10/18/2006)

Tremayne 2007 (attached to amendment filed 10/18/2006)

National Canine Cancer Foundation web site (<http://www.wearethecure.org/melanoma.htm>)

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Application No.: 09/996,128	Group Art Unit: 1642
Filed: 11/27/2001	Examiner: A Harris
Title: Compositions for Treatment of Melanoma and Method of Using Same	Confirmation No: 3698
Attorney Docket No.: MSK.P-026-3	
Customer No.: 52334	

DECLARATION UNDER RULE 132

I, Alan N. Houghton, declare as follows:

1. I am a named inventor of the above-referenced application. As such, I am familiar with the application, including the claims.
2. I understand that in the office action mailed December 27, 2006, the Examiner has cited Zhai et al., *J. Immunol.* 156: 700-710 (1996) as "teaching a method of inducing specific T cell immunity for mammalian metastatic melanoma."
3. The Zhai reference performs tests using B16 melanoma. B16 melanoma is not per se metastatic, and the parental B16 melanoma cells rarely metastasize. Classic experiments with B16 are described in the attached paper (1978). An important point about metastasis is that this represents multiple steps: motility and invasion of cancer cells into tissues, access into bloodstream or lymphatic vessels, spread through vessels to distant sites, migration out of blood vessels and survival in a distant, generally inhospitable tissue. This last point is underappreciated. If you inject 5 million B16 melanoma cells into the bloodstream from the parental B16 melanoma tumor, you will rarely get

metastases (the cells do not survive as lung metastasis, even though large numbers are trapped in the lung capillaries).

4. The Zhai paper only reports B16 melanoma, and do not described in metastatic version. In their experiments, B16 tumor cells are implanted in the skin, and the investigators look for local tumor growth. So-called metastases experiments typically use intravenous injection of tumor cells and look at colonization of organs (e.g., lung). Even these metastases models do not recapitulate all the events for metastases, e.g., tissue invasion and movement into vessels for dissemination. For these reasons, purists call these organ colonization experiments, not metastases experiments. Neither is disclosed in the Zhai reference, and thus there is no showing of relevance to metastatic melanoma.
5. B16 melanoma is not a viable model for canine malignant melanoma (CMM). In addition to not being metastatic, B16 is a single tumor line which spontaneously arose in a mouse ~50 years ago, and happens to grow well when transplanted into syngeneic (e.g. twin) mice. It almost certainly did not arise from the epidermis or mucosa, but rather from melanocytes in hair follicles since C57BL/6 mice do not have melanocytes in the epidermis or mucosa. In contrast, in dogs and humans, melanomas do arise from the epidermis and mucosa. In dogs, aggressive melanoma (CMM) arises from mucosal sites. B16 mouse models do not recapitulate the natural steps in pathogenesis of melanoma in dogs or humans. Moreover, chemotherapeutic drugs which are active against B16 melanoma (e.g., Stephens Br J Cancer 45:821-829, 1982; Stephens & Peacock Br J Cancer 38:591-598, 1978) are inactive in the treatment of melanoma (Houghton et al., Cancer Treat Rep 65: 170-171, 1981; Amrein, Am J Clin Oncol 7: 269-71, 1984.)

I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States

Code and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

dated: 2-21-2007

Alan Houghton  
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**Note:** Performing your original search, "*canine malignant melanoma*", in PubMed will retrieve 12 citations.

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1: J Vet Intern Med. 1999 May-Jun;13(3):163-74.

Related Articles. Links

## The molecular basis of canine melanoma: pathogenesis and trends in diagnosis and therapy.

**Modiano JE, Ritt MG, Wojcieszyn J.**

Department of Pathobiology, Texas A&M College of Veterinary Medicine, Texas A&M University, College Station 77843-4467, USA. jmodiano@cvm.tamu.edu

Melanoma is a common neoplastic disease of dogs with variable presentation and biological behavior. Canine malignant melanoma is a rapidly metastatic disease that generally is incurable. The loss of function of cellular safeguards built into the genetic program and of immune surveillance systems that cooperate to prevent tumor formation and progression appear to be important underlying causes of canine malignant melanoma. In effect, many existing cancer treatments restore the function of 1 or the other of these mechanisms. For example, chemotherapy and radiotherapy often kill tumor cells by initiating a genetic suicide mechanism (apoptosis), and immunotherapy initiates or enhances a response by the body's immune cells to identify and destroy cancer cells by mechanisms that rely on direct cytotoxicity or apoptotic cell death. Nevertheless, standard therapeutic approaches have not proved effective in treatment of canine malignant melanoma, with only marginal improvement in the outcome of dogs with this disease. The advantages of an improved understanding of the molecular basis of canine cancer are underscored by recent promising advances in diagnosis and in immunologic and genetic therapies that may help reduce the mortality of dogs affected with malignant melanoma.

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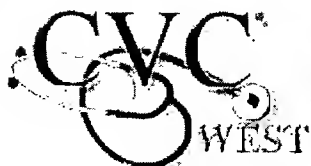
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## UC-Davis studies malignant melanoma in dogs

Aug 1, 2006

By: Jessica Tremayne  
DVM Newsmagazine

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**DAVIS, CALIF.** — Dr. Michael Kent, an investigator at the University of California-Davis, is exploring different drugs to help fight canine malignant melanoma.

The Morris Animal Foundation is providing a portion of the grant money necessary to conduct the research, which could unveil a way to make these types of tumors less resistant to treatment.

The fatality rate of this cancer is very high despite aggressive treatment with surgery, radiation therapy and chemotherapy.

"This type of cancer is fairly common and often fatal," Kent says. "We are testing drugs in cell lines developed from dogs with oral melanomas in combination with radiation."

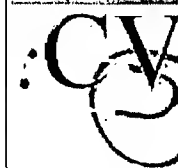
The tumors often are found in dogs' mouths, nail beds or skin.

Seizures and difficulty breathing can occur if the cancer spreads.

Cancer of the oral cavity accounts for 6 percent of canine tumors, making it the fourth most-common neoplasm in dogs, and malignant melanoma accounts for 30-40 percent of all oral lesions, Kent says.

"Similar to the disease in people, malignant canine melanoma is a highly metastatic tumor with many patients not surviving more than six months post diagnosis," he adds. "Surgery in the oral cavity for melanomas often requires extensive procedures, such as maxillectomy, mandibulectomy and/or orbitectomy and is rarely used for lesions located in the caudal portion of the mouth due to a substantial increase in morbidity.

Even with radical surgical procedures, local recurrence rate ranges from 25-43 percent. With radiation therapy, up to 83 percent of dogs having a complete or partial response,



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the time to regrowth or metastasis is often short lived with reported overall survival times of seven months.

Kent says if this work is successful, the next step will be to try these drugs in combination with radiation therapy.

The foundation is sponsoring the research for two years. Kent, in his sixth month of research, hopes the end result will improve the efficacy of treatment and lead to better tumor control.

#### About the Author

### Jessica Tremayne

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### DOG CANCER

Cancer is an uncontrolled growth of abnormal cells on or within the body. Cancer may be benign or malignant. It may be localized or it may invade adjacent tissue and spread throughout the body. The first step to preventing cancer is awareness and early detection. The National Canine Cancer Foundation and members of the Scientific Advisory Board has put together the following information on Cancer.

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### **Melanoma**

Melanoma occurs commonly in dogs with pigmented (dark) skin. Melanomas arise from pigment producing cells called melanocytes, which are responsible for coloring the skin. A dog can be affected, but Gordon Setters, Standard and Miniature Schnauzers, Doberman Pinschers, and Scottish terriers, among others, are at increased risk to develop melanoma, suggesting that this disease may have a hereditary component. Melanomas can occur in areas of haired skin where they usually form small, dark (brown to black) lumps but can also appear as large, flat, wrinkled masses. Melanoma of the haired skin in dogs is usually a benign tumor, although it can cause severe discomfort. In contrast, malignant melanoma, which develops in the mouth or in the distal (usually the toenail beds), is an incurable disease. These tumors have very often spread to distant parts of the body (metastasized) by the time they are first noticed, making complete surgical removal impossible.

Radiation therapy can help extend the lives of affected dogs but also is ineffective against tumor cells that have metastasized. Chemotherapy is also not considered capable of adequately controlling canine malignant melanoma.

Melanoma seems to be uniquely responsive to immune therapies, and various novel approaches are under development to treat this disease.

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